Cardiac Rehabilitation Manual

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Editor

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Preface

Embarking on a life-long journey

Cardiac diseases are still the leading causes of death in industrialized countries. They also induce considerable harm to survivors and often lead to severe and irreversible physical and neurological disabilities. Despite the fact that there is no cure, a lot can be done to prevent coronary artery disease, i.e., primary prevention, or to slow the progression of the disease, i.e., secondary prevention. Both can be achieved by tackling the panoply of modifiable risk factors, which have been identified to be amenable to lifestyle changes.

As a matter of fact, according to current guidelines, a long list of risk factors ought to be treated first by lifestyle changes before medical therapy is considered or initiated. These risk factors include:

- Physical inactivity
- Smoking
- Hypercholesterolemia
- Hypertryglyceridemia
- Low HDL cholesterol
- Arterial hypertension
- Hyperglycemia

As an example, physical inactivity has been recognized to be among the strongest predictors of morbidity and mortality for both otherwise healthy persons as well as already affected patients.

Often, however, medical therapy has to be initiated concomitantly to avoid further vascular damage and thus to halt or slow the progression of atherosclerosis. All doctors have received excellent training in choosing the right medication for their patients. We even have sales representatives from companies approach us on a regular basis who further try to provide as with up-to-date information. The only effective treatment that no one is offering us or our patients is exercise training. Neither do we receive information on dieting. We, thus, have to set out to try and find current and reliable information ourselves; an obvious deficit that this book is trying to reduce.

At the time that we start medical therapy we also tell our patients to change their lifestyle. But what exactly does this mean? What are the lifestyle changes that they are now being expected to make? And above all, can we provide our patients with an infrastructure that really helps them to deviate from current unhealthy behavior? Now it not only becomes very demanding for our patients but also for us, which is why many doctors, as shown in the EUROASPIRE trials, do not even recommend lifestyle changes; many because they lack detailed information on how to implement it.

Such training however is required, since it is not easy to convince a patient to say good-bye to many of his or her lifetime treats. Indeed, the vast majority has been leading an unhealthy lifestyle all their lives and may not wish to change this. If at all, it is right after a cardiac event that patients are amenable to our advice. This is the time to initiate changes that nobody can afford to miss. At the same time, these changes have to be agreed on with a patient who we see as partner on a lifelong journey of lifestyle changes, since otherwise patients may not necessarily stick to their good intentions in the long run. They need encouragement but also an infrastructure that ought to be available to them to actually modify their lifestyle.

Indeed, all our countries lack out-patient cardiac rehabilitation facilities that would provide a convenient and adequate infrastructure for our patients to not only initiate but also to provide a base for lifelong compliance with current guidelines. Such facilities have to be close to home, since otherwise it is not possible to attend exercise, nutritional, psychological, and other classes several times a week for an extended period of time. Only then, however, can long-lasting lifestyle changes be introduced into our patients' daily lives. Such facilities are especially warranted for those who want to return to work and wish to be on sick leave for as little as possible.

The network of institutions of ambulatory cardiac rehabilitation facilities has to increase, but also general hospitals have to start to establish ambulatory rehabilitation programs, so that patients get a fair chance to actually change their lifestyle. It is not enough if hospitals only concentrate on revascularizing patients, but do little or nothing to ensure optimal reduction in morbidity and mortality thereafter.

If we fail to do this, then we are in a situation that can be compared to prescribing drugs in a place where there are no drug stores.

But even if we were to get better infrastructure, even then we doctors have to improve our skills. Unfortunately, too few physicians have experience in cardiac rehabilitation, which comes as no surprise as it has never been taught in medical school, internist, or subspecialty training. It is only those of us who have chosen to work in cardiac rehabilitation centers or hospitals who know what to recommend and how to prescribe exercise training and other healthy treatment choices. I am no exception to this rule and had to learn the hard way by initiating training groups in various medical centers, what's best for our patients. Also several of the coauthors not only pursued a career in cardiac rehabilitation but got to where they are by trial and error. It is with this background and understanding that we hope to provide knowledge and advice to those who would like to learn more about cardiac rehabilitation.

After all it has becomes obvious that cardiac rehabilitation has not only come to stay but will become increasingly important, since it is a cost-effective treatment option.

As a matter of fact, the number and quality of cardiac rehabilitation programs have to increase, which in turn will require an increasing number of skilled staff. More doctors have to be trained adequately to receive the skills that are required to effectively recommend appropriate measures to patients, let alone to actually guide or accompany them on this lifelong journey. It is thus the aim of this book to provide doctors with in-depth but still hands-on information to quickly grasp the leading problems of our patients and to design or recommend appropriate programs. In this book, we have refrained from presenting exciting but exotic cases, but rather concentrated on the vast majority of our everyday patients in ambulatory or in-hospital cardiac rehabilitation.

All authors are members of the nucleus of the working group on cardiac prevention and rehabilitation of the European Society of Cardiology. Their expertise not only spans the whole spectrum of cardiac diseases but also contributes various aspects of challenges in cardiac rehabilitation from centers throughout Europe.

It is our wish to make a little but significant contribution to further excel the knowledge of our readers by writing this book which at first addresses general issues of cardiac rehabilitation, until it then teaches how to treat patients by focusing on individual patients with specific but very common cardiac conditions.

At first, this book will cover general principals of exercise testing and training as well as nutritional and psychological support. After these fundamentals of cardiac rehabilitation have been laid out in appropriate depths, chapters follow on the most common cardiac diseases. Cases include symptomatic coronary artery disease with or without diabetes, myocardial infarction or revascularization, and cases of heart failure in rather stable conditions, with or without cardiac devices. Our book will then be wrapped up with cardiac rehabilitation in patients with congenital cardiovascular diseases, valvular surgery, and peripheral arterial disease with claudication.

Contents is not presented in text book style, but rather taught on representative clinical cases. Each chapter focuses on a particular patient and discusses pros and cons of the most appropriate diagnostic tools and treatment options. It is thus designed to be a practical guide for doctors and geared to help them guide their patients.

Medical therapy, which most doctors will be very familiar with, has been addressed from the perspective of primary or secondary prevention and is of course in line with current guidelines of our national and international medical societies and associations.

A therapeutic option that has long and that still is terribly neglected will receive the attention that it deserves – physical exercise training. Data on reduction of morbidity and mortality but also on improvement in quality of life are that striking that neither we nor our patients can afford to not use this poly-pill. Most of the modifiable risk factors of cardiovascular diseases can be treated by these lifestyle changes. Nevertheless, in the real world, treatment strategies concentrate almost solely on pharmaceutical interventions, neglecting the beneficial effects of heart-healthy diets and exercise training programs. For managing both long- and short-term risk, lifestyle changes are the first-line interventions to reduce the metabolic risk factors. Indeed, the importance of physical activity and heart healthy nutrition cannot be overestimated. This will be highlighted in several chapters.

Primary and secondary prevention of cardiovascular diseases needs to focus on all modifiable risk factors and implement pharmaceutical therapy wherever appropriate.

Exercise training has to become an integral part of it. It is inacceptable that it is only integrated into the daily routine by a minority of patients. Further cardiac rehabilitation programs have to be installed and doctors need to be trained to be able to refer and treat patients at this stage in their disease history appropriately. We strongly believe that this book will add to the knowledge of our readers and that it will enable them to better guide their patients on a lifelong journey of primary and secondary prevention.

May 2010 Josef Niebauer, MD, PhD, MBA

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Part I

Introduction to Cardiac Rehabilitation

General Principles of Exercise Testing in Cardiac Rehabilitation

1

Miguel Mendes

1.1 Introduction

Before admission to a Cardiac Rehabilitation Program (CRP) every patient must be submitted to a clinical assessment which must include a medical consultation, an evaluation of LV function (usually by echocardiography), a maximal exercise test (ET) limited by symptoms and blood tests to evaluate CVD risk factor profile. In special cases, additional tests like 24 h Holter monitoring, stress echo, myocardial perfusion scan or even a coronary angiogram has to be performed.[1–5]

The ET is a very important part of this clinical assessment performed prior to admission and at the end of a CRP phase, because it gives indispensable data regarding functional capacity, hemodynamic adaptation to maximal and sub maximal levels of exercise heart rate (HR) and blood pressure (BP), residual myocardial ischemia, cardiac arrhythmias induced or worsened by exercise and allows the calculation of the training heart rate (THR) for the aerobic training.[2–4]

Besides the objective parameters mentioned above, the ET is very important psychologically for many patients and their partners, because they can realize that, after the cardiac event, the patient usually already has a better functional capacity than they could predict. In the follow up period, the ET is very useful to detect or confirm eventual clinical status changes that occur during the program, update exercise prescription intensity, measure the gains obtained after the CRP and for global prognostic assessment.

1.2 What Kind of Exercise Test?

Cardiopulmonary exercise test (CPX) is the ideal exercise test (ET) to be used in all types of patients in the setting of a CRP.[6] Although it is almost mandatory to use it in heart failure patients,[3] due to its higher cost, more complicated delivery and interpretation, it is

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usually replaced in many CRP centers, especially in CAD patients with normal or near normal LV function, by the standard ET which is widely available and familiar to most cardiologists.

During the CPX, peak VO_2, anaerobic threshold, VE/VCO_2 slope, and O_2 kinetics are measured in addition to all the parameters recorded in the standard ET, such as the maximal work load reached, the HR and BP changes from rest to maximal exercise and recovery, the eventual arousal of symptoms like angina pectoris or ECG abnormalities (ST changes or arrhythmias).[7]

Considering the parameters obtained from the CPX, peak VO_2 is the most important since it is the gold standard for functional capacity and it has been identified as the strongest prognostic parameter in CVD.[8–12] Peak VO_2 is also very important for determining optimal exercise intensity because exercise training must be performed at a percentage of peak VO_2 ranging from 50% to 70%.[13]

The anaerobic threshold (AT), which is expected to increase during the CRP, being independent of motivation, is considered a good indicator of the training effect.

1.3 How to Perform an ET in the Setting of a CRP?

A fully equipped ET set-up, including at least one type of ergometer (bike or treadmill), an ECG system with several exercise protocol options, an emergency cart, together with a well-trained and experienced staff (cardiologist and technician), must be available to perform an ET in the setting of a CRP.[7,14,15]

After welcoming the patient immediately before the ET, the patient must be asked about his exercise tolerance, in order to estimate his maximal functional capacity and enabling choice of appropriate test protocol. The test must be programmed in such a way that the patient's physical exhaustion or high grade fatigue will be attained at about 10 min of exercise.[16] In case of test interruption before 8 min, a less intense protocol must be used (with a steeper one in the reverse situation) to evaluate the functional capacity correctly.

Other questions that need to be asked are related to a possible recent worsening of clinical status, which may require the postponement of the test, and if the patient stopped taking his regular cardiovascular medication before the test day and time. ETs integrated in a CRP must be performed under the patient's usual medication and one must try to schedule the test at a similar time of day to that of the CRP sessions.

Ensuring that medication and timing are optimal for the test will mean that the drug effect and consequently the patient's protection will be different during ET and the exercise sessions of the CRP. This is very important, for example, in patients on beta-blockers, which are usually taken in the morning and whose effect can diminish during the day. If the THR was calculated after a test performed at a certain time of the day, it may be much harder to reach if the test was performed late in the day and the CRP session is early in the morning, where the beta-blocker effect is more intense or will be easily surpassed in late afternoon exercise sessions if the ET took place in the morning.

The decision to stop the exercise phase is crucial to quantify exercise tolerance accurately. If no medical contraindications to continue the effort are present, such as major ST changes, serious arrhythmias, BP drop, or hypertensive response, and if the patient seems to be relatively comfortable, the exercise period must be interrupted only upon patient request, based on the perception that he has reached his maximal exercise capacity or feels major respiratory discomfort, leg claudication eventually related to peripheral vascular disease, or orthopedic disease.[14,15]

The exercise period must not be stopped based on the attainment of any level of predicted maximal HR, due to the large variability of peak HR among subjects. This procedure prevents an accurate definition of exercise tolerance and maximal HR. The correct detection of maximal HR is very important to calculate the THR, which is based on the chronotropic reserve and VO_2 at the ventilatory AT (VAT) or at peak exercise level.

After confirming that the patient is suitable in terms of clinical status and medication, it is time to choose the ergometer and the protocol:

If both of the usual possibilities are available (bicycle or treadmill), the choice must be made taking into consideration the ergometer which will be mostly used for aerobic training, patient preference and experience of the clinical staff.

Regarding the protocol choice, two issues must be considered:

1. The patient predicted exercise tolerance
2. Type of protocol (ramp type or with small versus large increments between stages).

Considering the type of increments of the ET protocol, there is a preference for ramp or short increment (around 1 MET; metabolic units of oxygen consumption: 1 MET = 3.5 mL/kg/min) protocols,[17,18] because the error of the functional capacity estimation will be lower, in the case that respiratory gas analysis will not be performed. The ergometer must also be taken into consideration because the load is more accurately determined on a bike than with the treadmill, since the treadmill calibration is harder and the patient usually grasps the handrails during the effort, diminishing the oxygen demand needed to perform the test (Tables 1.1 and 1.2).[7,14,15]

Table 1.1 Stationary bike: most commonly used protocols[19,20]

Designation	Load (W)			Duration (min)		Peak estimated METs
	Start	Increase	Peak	Stage	Total	
Balke (men)	50	25	175	2	12	9.5
Balke (women)	25	25	150	2	12	8.3
Astrand	25	25	150	3	18	8.3

Table 1.2 Most commonly used treadmill protocols[21–23]

Designation	Estimated METs		
	At 8 min	At 9 min	At 12 min
Naughton	4	NA	6
Balke-Ware[a]	5	NA	8
Modified Bruce	NA	7	10
Bruce	NA	10	13

NA – not applicable

[a]Usually not acceptable for old people and frail patients, because it has a high constant speed (5.47 km/h), not tolerable for most of these patients

1.4 When to Do It

The ET must be performed on admission to the CRP in the majority of the cases, sometimes in the middle of a phase when it seems that the patient's clinical status has changed or THR is inadequate due to the acquisition of a better exercise tolerance as a consequence of exercise training, and at the end of each phase to measure the final functional capacity.[2,4,13]

Patients recently submitted to cardiac surgery usually start exercise training without performing an ET, because they may have physical limitations that require postponement of the test for 2–4 weeks. During these early weeks, the patients are involved in respiratory and global physiotherapy and may even start exercising on a stationary bike or on a treadmill, below a THR of 100 or 120, respectively, until they reach a satisfactory exercise tolerance that enables them to be submitted to the ET, after which time an individualized THR will be calculated.[2]

1.5 How to Report the ET in the Setting of a CRP?

A standard ET must be reported not only in terms of presence or absence of myocardial ischemia, but should also include overall prognosis, functional capacity, chronotropic index, HR recovery, BP, ventricular or supraventricular arrhythmias (Table 1.3).

Despite having informed the patient at the beginning of the ET about the need to spontaneously report the occurrence of any unexpected symptom, namely, angina or an out of proportion grade of dyspnea or fatigue, the patient must be asked periodically (for example at the end of each stage) and at the moment of ST depression occurrence if he is experiencing angina and what is his perception of exercise intensity (Borg scale). During the exercise period it is also recommended to record a full ECG every minute in order to define accurately the eventual moment at which ST segment depression reaches 1 mm, 60 or 80 ms after the J point, the so-called ischemic threshold.

Table 1.3 Parameters to describe in the ET report in the CR setting[15,24]

1. Exercise capacity (a) Test duration and reason to stop exercise (b) Quantification of exercise tolerance, as ratio of the achieved and the predicted METs, calculated by the following equations: (i) Men: Predicted METs = 14.7 − 0.11 × age (ii) Women: Predicted METs = 14.7 – 0.13 × age Classify functional capacity below normal if lower than 85% of the predicted value
2. Heart rate – HR at rest, at the end of each stage, at the moment of the ischemic threshold, ventricular or supraventricular arrhythmias, abnormal BP (i.e. drop in BP or hypertensive response, at peak exercise and in recovery at 1, 3 and 6 min) Classify chronotropic evolution during exercise as: – *Normal*, if peak HR value is above 85% of the predicted value (220 bpm – age), for individuals not under β-blocker or above 62% under β-blocker – *Abnormal*, if below the mentioned values Classify chronotropic evolution during recovery as: – *Normal*, if HR difference between peak exercise and min 1 is > 12 bpm on protocols with an active recovery (slow walking or pedaling) or > 18 bpm, if exercise is immediately stopped at peak effort – *Abnormal*, if below the mentioned values
3. Blood pressure Classify blood pressure evolution as: – *Normal*, if SBP increases ~10 mmHg per MET and there is no change or a small drop is found in DBP. It's acceptable to find a drop <15 mmHg at peak exercise on SBP – *Hypertensive*, if SBP reaches values >250 or > DBP 120 mmHg – *Insufficient*, if SBP increases <30 mmHg
4. Ischemia – Classify the test as *negative*, *positive*, *equivocal* or *inconclusive* for myocardial ischemia, taking into consideration the presence or absence of angina or ST depression/elevation induced during the test, in the exercise or the recovery period, according to the criteria defined in the guidelines – Use the ST/HR index, the ST rate-recovery loops and/or the ST/HR slope to increase the accuracy of the diagnosis of ischemia – Grade ischemia as severe, moderate or mild, taking into consideration the appearance and the magnitude of ST changes, the time until normalization in the recovery period, the association with limiting angina, BP fall, chronotropic deficit or ventricular arrhythmias – Identify clearly the HR of the ischemic threshold, because the THR to be observed during the exercise sessions must be 10 bpm below this value for safety reasons
5. Prognosis Assess globally the prognosis, considering functional capacity, ST/HR index, chronotropic response, HR recovery, ventricular ectopy during recovery and ST/HR slope, which are implicated in predicting global and cardiovascular mortality and events
6. Aerobic training intensity Calculate the THR (training heart rate) as the HR at (50), 60–70% of HR reserve or (50), 60–70% of VO_2 reserve or at HR of the VAT level, respectively, if the patient was submitted to a standard ET or to a CPX

Ischemia is diagnosed by the occurrence of angina and/or definitive ST changes on exercise or in the recovery period. In order to increase the diagnostic accuracy of the ET, ST changes must be interpreted considering ST/HR index, which must be over 1.6 μV/bpm, and rate recovery loops (that are suggestive of myocardial ischemia if there is a counterclockwise rate-recovery loop).

Functional capacity is probably the most important finding of an ET, as it is the best parameter to predict all-cause mortality. When peak VO_2 is not measured, it can be estimated by the ratio between the achieved METs calculated from the last stage of the ET protocol metabolic demand and the predicted value given by the following formula: Predicted METs = 14.7 – 0.11 × age or 14.7 – 0.13 × age, respectively, for men and women.

The Duke score tries to put together the presence/absence of ischemia and functional capacity, and classifies the patients into low, intermediate, and high categories of risk, according to the value of the score.

Chronotropic index, HR recovery and ventricular arrhythmias predict increased/decreased risk of death if they are negative or positive.

1.6 How to Assess Exercise Training with a Standard ET or a CPX?

At the end of a CRP phase, the ET or the CPX must be repeated to be compared with the test performed at the phase start, in order to document eventual gains provided by the program.

These gains must be observed in terms of maximal and sub maximal functional capacity, ischemic threshold, exercise induced or worsened arrhythmias, heart rate and blood pressure evolution during the exercise and the recovery periods.[2,6,13]

To make a correct comparison between both tests, they must be performed under the same medication, at the same time of the day, using the same ergometer and protocol. If any revascularization procedure, like a percutaneous coronary intervention (PCI), is performed, the medication is changed, the ergometer or the protocols are different, a direct comparison of both tests is impossible, namely if a standard ET was performed. If both tests were a CPX, even with different protocols, it is possible to compare functional capacity.

1.6.1 Standard ET

If exercise training is successful, the second ET will usually show:

(a) Higher duration/load attained
(b) Lower levels of HR and BP at each stage and an earlier normalization of HR during recovery

(c) Starting of ischemia later during the test, usually at the same double product
(d) Lower frequency and complexity of ventricular arrhythmias,

Functional capacity can be estimated for each patient in terms of METs, considering the oxygen consumption previously known to be inherent to the highest stage attained at peak exercise, if the patient was able to workout at this stage for more than 1 min. If the test was stopped before 1 min, the attributed estimated METs must be those predicted for the previously completed stage.

Functional capacity must also be classified by considering the predicted values for the same age, gender and physical activity status, which are provided by several equations.

The maximal load reached by the patients, can also be considered as a measure of functional capacity, especially when a stationary bike is used. On a treadmill, due to the body weight dislocation effect, the peak load values are less accurate.

The estimation of aerobic capacity by the standard ET is not very accurate, since it usually overestimates the load, namely, in the presence of cardiac disease, advanced age and when high increment treadmill protocols are used, like the Bruce protocol.

1.6.2 Cardiopulmonary Exercise Test

The CPX allows the best identification of maximal aerobic capacity because peak VO_2, the gold standard for exercise capacity, is directly measured "breath-by-breath" during the entire test. Due to some variability, the values should be determined by calculating the rolling average of each period of 30 s.[25]

Peak VO_2 is the most used parameter to evaluate the CRP benefit. If there is doubt that the CPX is a maximal test, one must specifically look at VO_2, HR and respiratory exchange ratio (RER), and rate of perceived exertion (RPE) at peak exercise level. While VO_2 and/or HR must fail to increase significantly despite a further increasing load, RER and RPE must be at least 1.10 and 8/10 at peak.[26]

To overcome the limitations of peak VO_2, the VO_2 attained at the VAT can be used to evaluate the training effect, because it is independent of patient's motivation and expresses better the patient's capacity to perform daily life activities.

In cardiac patients, peak VO_2 and VO_2 at VAT increase between 7% and 54% after a period of some weeks of exercise training, although the average increase is usually around 20 to 30%.[27–29]

VE/VCO_2 slope, which evaluates ventilatory efficiency, one of the most important parameters for prognosis assessment in CHF, is also expected to decrease as a demonstration of a favorable exercise training period (Table 1.4).[30]

Table 1.4 How to assess the training effect with an exercise test[7,14,15]

Standard ET	Cardiopulmonary exercise test
• Test duration, maximal load and estimated METs • Presence or absence of ischemia • HR at rest, in each stage, at peak exercise and during recovery • Blood pressure at rest, in each stage, at peak exercise and during recovery • Ischemic threshold: HR, double product and load • Grade of myocardial ischemia, in terms of ST normalization, ST depression morphology • Ventricular arrhythmias	The same parameters as in standard ET, plus: • Peak VO_2 • VO_2 and HR at VAT • O_2 kinetics in the recovery period • Peak RER • VE and breathing reserve • VE/VCO_2 slope

1.7 Clinical Cases

Case #1

Male, 41 years old

Apparently healthy man very recently suffered an anterior myocardial infarction. Tobacco smoking and obesity were identified as risk factors for CVD in this case: he smoked one pack a day in the last 25 years and has a BMI of 30.6 (99 kg weight and 180 cm height). His BP, blood cholesterol and glucose levels were normal. Prior to his infarct he was under considerable psychological stress.

He was submitted to a primary PCI of the LAD (middle portion), that was totally occluded by a thrombus. The PCI was performed within 2 h of symptoms and was successful, with the exception of the occurrence of a right thigh hematoma related to the femoral puncture, which obliged him to stay in bed for a week. No other lesions were found in the coronary arteries and LV function was near normal.

He was discharged from the hospital on the fifth day after ACS under ASA, clopidogrel, ramipril (2.5 mg, od), bisoprolol (2.5 mg, od) and pravastatin (40 mg, od).

When he started to get out of bed and to move around 1 week after hospital discharge, he felt dizziness, nausea and a thoracic discomfort, different to the one that arose during the ACS, which stopped immediately when he laid down. No pericardial effusion was found on echocardiography. After this he took the initiative to contact our CRP 3 weeks after the ACS.

After a medical consultation and physical examination, where everything seemed to be OK he was submitted to an ET (Table 1.5, Fig. 1.1):

Table 1.5 Exercise test parameters (case #1)

Stage	Speed (km/h)	Grade (%)	METs	HR (bpm)	SBP (mmHg)	DBP (mmHg)	Symptoms	ECG
Rest	0	0	1	75	130	80	No	Normal
I	2.7	10	4.6	98	150	80	No	Normal
II	4.0	12	7.0	117	175	90	Mild fatigue	Normal
III	5.4	14	10.0	138	200	100	Moderate fatigue	Normal
IV	6.7	16	12.5	150	210	100	Severe fatigue	Normal
Exercise duration: 10 min 20 s								
Rec. 1′	1.5	0		132	200	90	No	Normal
Rec. 3′	0	0		104	190	90	No	Normal
Rec. 6′	0	0		95	170	85	No	Normal

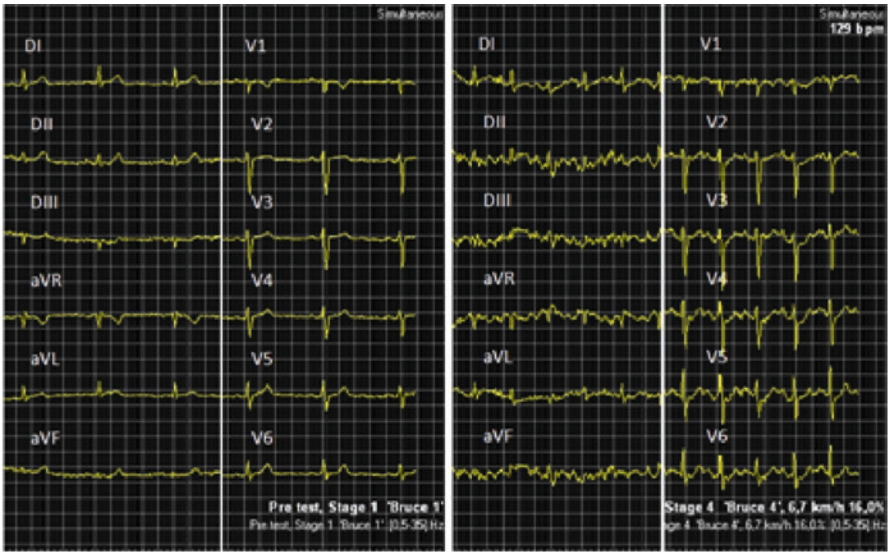

Fig. 1.1 Rest and peak exercise 12-lead ECG (case #1)

Comments ET#1

Confronting the findings of this ET with what is supposed to be found in a normal ET, this patient shows:

1. Good exercise tolerance: 10:20 min exercise duration on the Bruce protocol ~ 12.5 METs (122% of the predicted).
2. Normal evolution of HR: from 75 to 150 bpm at peak effort with a drop of 18 bpm on the first minute of an active recovery.
3. Normal increase of SBP: from 130/80 at rest to 210/100 at peak exercise.
4. Hypertensive pattern on DBP: increase from 80 to 100 mmHg
5. No arrhythmias, ST changes or angina.

Comments

This is a typical case of a low risk patient for a CRP, with normal LV ejection fraction, no residual ischemia, no arrhythmias, good exercise tolerance and a normal adaptation of hemodynamic parameters to maximal exercise.

He was admitted to a formal CRP under medical supervision, since he wished to enrol on an exercise program.

A THR of 120 bpm was calculated using the Karvonen formula, adding 60% of his HR reserve [(150–75)*0.60 = 45 bpm] to the rest HR (75 bpm): 45 + 75 = 120 bpm[31–34].

Case #2

Male, 54 years old

CVD risk factors: Type 2 diabetes and hypertension.

Assessment performed before admission to CRP on the 4th October 2004, following a non complicated CABG on 11th July 2004 and a previous inferior myocardial infarction of no definite date.

Submitted to complete revascularization, by a: triple CABG with LIMA to LAD and single saphenous graft to the second diagonal and posterior descendent arteries. Three months after surgery, a nuclear perfusion scan, requested for routine clinical assessment, identified residual silent ischemia in the inferior wall (Fig. 1.2).

After this test he was re-submitted to coronary angiography where it was found that the graft to the posterior descendent artery was occluded and the artery was not considered to be amenable to PCI. He had good collateral circulation from the left coronary artery and the other bypass was patent with normal flow. His attending cardiologist decided to keep him on medical therapy and send him to the CRP.

Before CRP he was submitted to an ET, under his usual medication: bisoprolol (5 mg, od), nitrate (50 mg, od), losartan (50 mg, od), enalapril (20 mg, od), thiazide (12.5 mg, od), sinvastatin (20 mg, od), aspirin (100 mg, od) and two oral antidiabetic drugs (Table 1.6).

After 12 weeks of CRP, he was re-assessed by a new ET, under the same protocol (Bruce) and medication (Table 1.7).

Comments

First test:

The patient had residual ischemia with a moderate compromise of functional capacity (seven estimated METs). He was admitted to the CRP, with a THR of 100 bpm, calculated by the Karvonen formula, adding 60% of his HRR to the rest HR: [(131–59) × 0.60] = (43 + 59) = 102 ~ 100 bpm. If the calculated value for THR would be superior or equal to the HR of the ischemic threshold, which was 123 bpm in the exercise test on admission (Table 1.6) the THR would be assigned to a HR 10 bpm lower than the HR of the ischemic threshold, what would be around 110–115 bpm

He didn't complain about any symptoms during the program and he progressed very well.

Second test:

The second test was performed immediately upon completion of the program. It showed a very good evolution[35]:

1. Better functional capacity (12.5 vs 7.0 estimated METs)
2. Lower values of HR at each stage of the protocol, with silent ischemia appearing almost at the same HR value, although much later in the ET. Although some references show that ischemic threshold can appear at a higher HR and double product after exercise training, in this case, as usual, only a delayed appearance was found.
3. Higher HR and BP values at peak exercise.

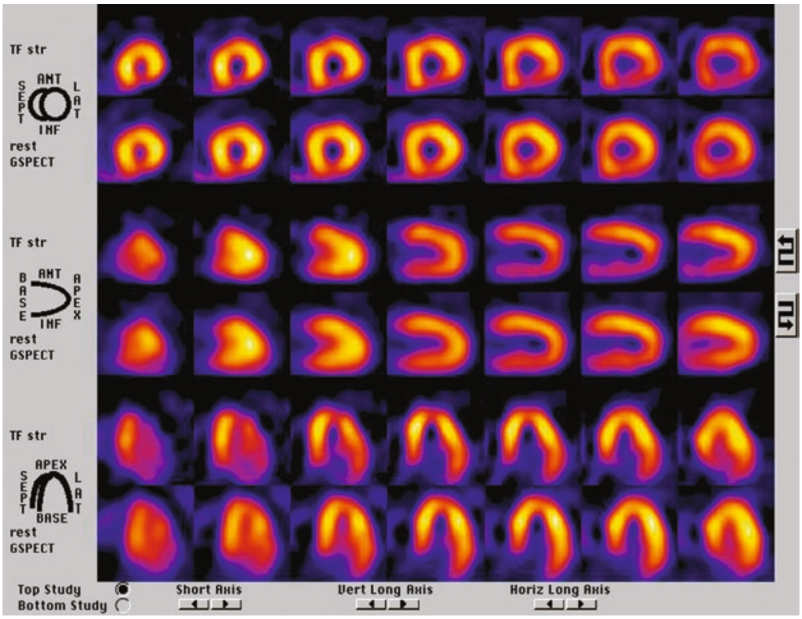

Fig. 1.2 Myocardial nuclear perfusion scan (case #2)

Table 1.6 Admission exercise test (case #2)

Stage	Speed (km/h)	Grade (%)	METs	HR (bpm)	SBP (mmHg)	DBP (mmHg)	Symptoms	ECG
Rest	0	0	1	59	130	80	No	Q waves in II, III and aVF
I	2.7	10	4.6	113	150	80	No	No change
II	4.0	12	7.0	131	170	80	Intense fatigue	ST downslope of 1 mm in V5–V6
Exercise duration: 6 min 00 s; onset of ischemia: at 4 min 00 s with 123 bpm								
Rec. 1′	1.5	0	1	112	140	80	No	ST downslope of 1 mm in V5–V6
Rec. 3′	0	0	1	90	130	80	No	ST downslope of 1 mm in V5–V6
Rec. 6′	0	0	1	82	120	75	No	ST downslope of 1 mm in V5–V6
Rec. 9′	0	0	1	79	120	80	No	Equal to rest ECG

Table 1.7 End of CRP exercise test (case #2)

Stage	Speed km/h	Grade%	METs	HR bpm	SBP mmHg	DBP mmHg	Symptoms	ECG
Rest	0	0	1	68	120	90	No	Inferior Q waves
I	2.7	10	4.6	91	160	80	No	No change
II	4.0	12	7.0	103	170	80	No	No change
III	5.4	14	10.0	125	190	80	Mild fatigue	ST downslope of 1 mm in V5–V6
IV	6.7	16	12.5	142	190	80	Intense fatigue	ST downslope of 1 mm in V5–V6

Table 1.7 (continued)

Stage	Speed km/h	Grade%	METs	HR bpm	SBP mmHg	DBP mmHg	Symptoms	ECG
Exercise duration: 10 min 00 s; Onset of ischemia: at 10 min 00 s of exercise with 123 bpm								
Rec. 1′	1.5	0	1	123	190	80	No	ST downslope of 1 mm in V5–V6
Rec. 3′	0	0	1	95	180	80	No	ST downslope of 1 mm in V5–V6
Rec. 6′	0	0	1	88	130	80	No	ST downslope of 1 mm in V5–V6
Rec. 9′	0	0	1	80	120	75	No	Equal to the rest ECG

Case #3

Male, 64 years old, asymptomatic, submitted to ET on 12.03.2009, 5 days after inferior STEMI;

Risk factors: dyslipidemia, hypertension, smoking and family history of CVD below 60 years.

Medication: aspirin, clopidogrel, ß-Blocker, ACE inhibitor and statin

Baseline ECG: sinus rhythm; Q waves on inferior leads

Coronary angiography: left main lesion < 50%. Occlusion of RCA on the middle portion, with retrograde filling from the left coronary. No significant lesions were found on LAD and circumflex arteries (Table 1.8, Figs. 1.3 and 1.4).

Summary

METS = 4,6 (59% of the predicted)

Peak HR: 113 ppm = 72% predicted HR;

% HR reserve use: 72.4% (abnormal if ≤ 62%)

HR decay in the first minute of recovery: 11 bpm (abnormal ≤ 12 ppm)

Peak double product = 16,950

ST changes: Horizontal downslope ST segment depression starting at 3 min of exercise, with a maximal amplitude of 1.5 mm in V4, V5 and V6. ST depression normalized at 9 min of recovery, after sublingual nitroglycerine at 6 min.

Arrhythmias: absent

Table 1.8 Admission exercise test (case #3)

Stage	Speed km/h	Grade %	METs	HR bpm	SBP mmHg	DBP mmHg	Symptoms	ST-T changes
exercise data								
Rest	0	0	1	75	120	60	None	
I	2,7	10	4,6	111	150	80	Fatigue	ST depression ST = 1.0 mm
II	4	12	7	113	150	80	Fatigue, not-limiting angina	ST depression ST = 1.5 mm

Test stopped at 3 min and 33 s of Bruce protocol due to fatigue

Recovery data

Stage	HR (bpm)	SBP (mmHg)	DBP (mmHg)	Symptoms	ST-T changes
Rec. 1′	102	150	80	None	ST depression ST = 1.5 mm
Rec. 3′	90	140	80	None	ST depression ST = 1.5 mm
Rec. 6′	78	120	70	None	ST depression ST = 1 mm
Rec. 9′	81	120	70	None	Absent

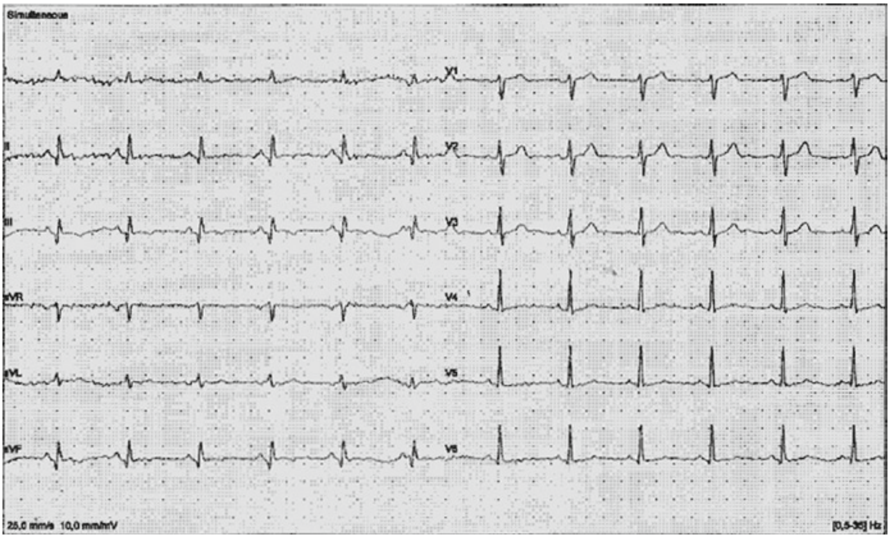

Fig. 1.3 Rest 12-lead ECG (case #3)

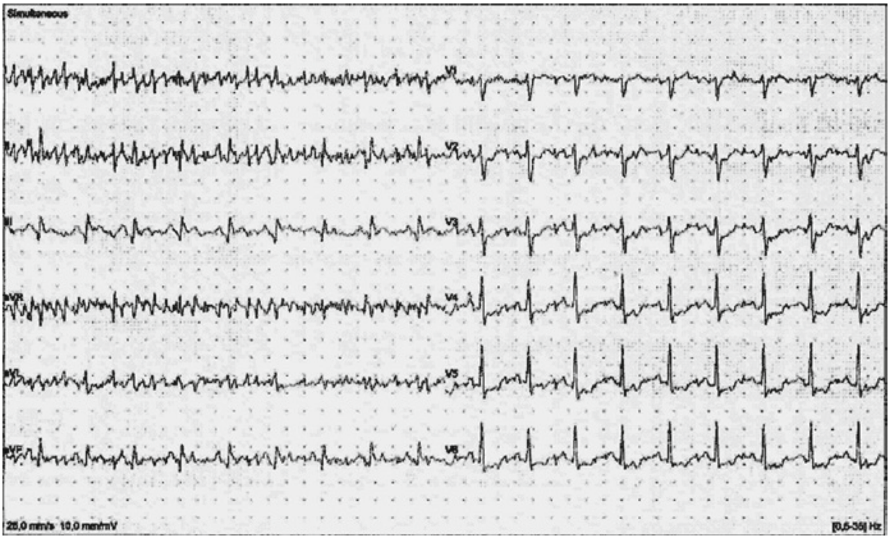

Fig. 1.4 Peak exercise 12-lead ECG (case #3)

Conclusions

1. Moderate compromise of exercise tolerance (<5 METs).
2. Myocardial ischemia starting at low exercise level (see Table 1.9)

Comments

The patient was not accepted in the CRP, since he had myocardial ischemia starting below five METs, a contraindication for admission.

This patient's myocardial ischemia must be considered serious, since it starts at low level of exercise, is associated to angina (although non-limiting) and normalized only at 9 min in the recovery period after sublingual TNG.

He was submitted for a new coronary angiography where IVUS was performed. The left main lesion, with an area of 10.4 mm^2 and a plaque burden of 47% on IVUS, was considered as non-significant (Fig. 1.5), but a lesion of 70% of the first marginal obtuse was defined as the lesion responsible for the ischemia. This lesion was submitted successfully to PCI with drug-eluting stent insertion.

He was re-evaluated and admitted to the CRP 1 week later, after being submitted to a new ET that showed good exercise tolerance (9 min on Bruce protocol) and no residual ischemia.

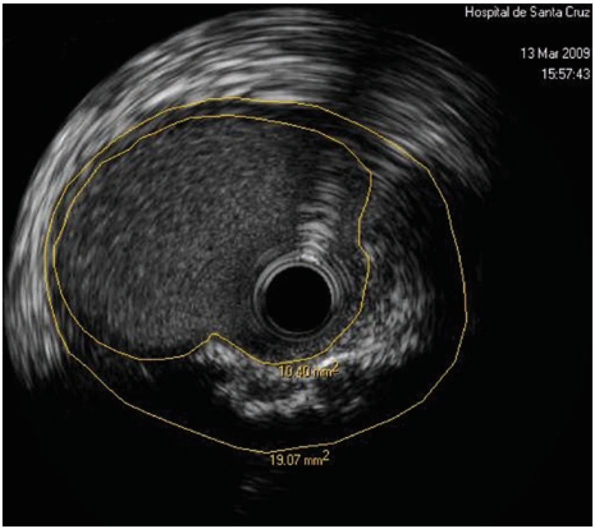

Fig. 1.5 Left main IVUS (case #3)

Table 1.9 Risk classification grades for exercise training in cardiovascular patients (Adapted from refs.[2] and[14])

Risk	Low Stable conditions	Moderate	High Unstable conditions
Decision regarding patient admission in the CRP	Accept	Decide case by case.	Consider to reject or return to referral MD for stabilization
Type of CRP team	Basic experience	Advanced experience	
Individualized exercise prescription	Yes	Yes	Exercise training recommended only in few specific situations
Sessions supervision	Nonmedical personnel with Advanced Cardiac Life Support	Medical and nonmedical with Advanced Cardiac Life Support, until safety apparently guaranteed	As in moderate risk patients
ECG and BP monitoring	6 – 12 sessions	≥12 sessions	≥12 sessions
NYHA	I or II	III	IV
Exercise capacity	≥7 METs	<5 METs	<5 METs
Myocardial ischemia	Absent or ≥7 METs	<7 METs	<5 METs
Ejection fraction	≥50%	40–49%	<40%
Rise of BP and HR	Appropriate	Appropriate	Fall or non-increase of SBP or HR during exercise
VT at rest or during exercise	Absent		Complex arrhythmias
Self-monitoring	Able	Some difficulties	Unable